A PARASITE RESEMBLING *PLASMODIUM FALCIPARUM* IN A CHIMPANZEE

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PLATE IX

The following observations were made by us on a chimpanzee, *Anthropopithecus troglodytes*, at Freetown, Sierra Leone. The animal, according to the statement of the owner, had suffered from an attack of dysentery lasting from January 1st to January 16th, 1922. It was examined by us on the 11th and 12th of January, at which time it was passing blood, pus and mucus. The only organisms found in the faeces by microscopical examination were large numbers of *Blastocystis* resembling *Blastocystis hominis*; no bacteriological examination of the faeces was made; the cutaneous blood was examined, and no parasites were found. From January 16th to January 31st the animal was well. On January 31st and February 1st it refused food; its condition improved on the 2nd and 3rd, but the next day it became worse, and the owner handed it over to us. The chimpanzee was very thin, its hair was coming out and it was obviously ill; on February 5th it had an attack of diarrhoea; no blood was passed; *Strongyloides* larvae were present in large numbers in the faeces.

Malaria parasites were found in the blood on February 4th. They increased in number and the animal's condition became worse. On the 8th of February, in the afternoon, it was somnolent, and on the 9th it refused food and drink, lay motionless, was not easily

roused, and remained in any attitude in which it was placed without attempting to change it. At 8.30 a.m., after considerable retching, it vomited. As the animal's condition was grave and appeared to be associated with the increasing number of parasites in its blood, 5 grs. of quinine bisulphate in solution were administered orally at 10.30 a.m. From February 11th till 19th its condition improved; the number of parasites in the peripheral blood was reduced rapidly by the action of the quinine, but the blood was never free. On the 19th the blood was again heavily infected, more so than on any previous occasion; the animal was ill, but its condition was not as grave as it was on the 9th, and by the 21st its appetite returned and it began to recover without any quinine. On the 22nd the animal was lively and eating well, and the parasites in its peripheral blood were decreasing. On February 23rd, at 8.30 a.m., the animal appeared well and made a good meal. At noon the same day it was found lying in its cage in a condition of collapse and breathing with difficulty; it had vomited a large quantity of bile-stained material. Death occurred in half an hour.

Post-mortem examination. The immediate cause of death appeared to be innumerable small haemorrhages which were uniformly distributed over the whole surface of both lungs; these haemorrhages were very recent, and on examination proved to be caused by the presence of Strongyloid larvae. An account of the changes produced by the larvae and the sites in which they were found will be given in a future communication. The trachea contained a small quantity of regurgitated food, but this was not sufficient to cause obstruction. The vessels on the surface of the brain were dilated; there were no haemorrhages on the surface or in the substance of the brain; there was no meningitis. The spleen was not greatly enlarged; it was very dark in colour and somewhat harder than normal. The liver was dark and congested. The bone marrow was dark red. The kidneys and the heart appeared normal.

EXAMINATION OF SMEARS AND SECTIONS OF THE ORGANS

Brain. A few trophozoites and gametocytes were found; the capillaries were not blocked with parasites; pigment was present in small amount.

Spleen. Trophozoites and schizonts were found, but were not numerous; masses of pigment were present; there was considerable fibrosis.

Liver. This contained pigment in very large amount; it occurred in granules and in coarse masses; some of the smaller granules were found in the liver cells.

Bone marrow. Trophozoites and gametocytes were present, and coarse pigment was plentiful.

Blood. Trophozoites and gametocytes were present, but were not very numerous; very heavily pigmented leucocytes were common.

TYPES OF PARASITE FOUND IN THE BLOOD

1. Large amoeboid trophozoites resembling *P. vivax*, in pale enlarged red cells.

2. Large heavy looking trophozoites more or less band-shaped and equatorial, coarsely pigmented, resembling *P. malariae*.

3. Trophozoites resembling small rings of *P. falciparum*. The red corpuscles were not enlarged and retained their colour.

4. Gametocytes were found, indistinguishable from those of *P. falciparum*; they were never present in large numbers throughout the course of the disease.

No schizonts were found in the blood.

The *P. vivax* and *P. malariae* forms were scanty; they were found on the 4th and 5th of February, but were not seen subsequently. After the 5th of February the parasites seen were invariably of the *P. falciparum* type; they showed a certain amount of pleomorphism, but this was not more notable than in the case of the human parasite. The pleomorphism consisted in the appearance of slightly amoeboid and *tenue* forms. Crescents appeared in largest numbers on the 12th and 13th of February, but even then were not numerous; no exflagellating forms were found.

IDENTITY OF THE PARASITE

The few parasites of the *P. vivax* type corresponded to *P. inui*, Halberstaedter and Prowazek, 1907, in that the host cell was enlarged and pale, and did not present Schüffner's dots. As they and the *P. malariae* forms were not found in the blood on or after

the 6th February, the conclusions drawn from the experiments detailed below cannot be considered strictly applicable to them.

Reichenow (1920) records the discovery of parasites identical morphologically with *P. falciparum* in chimpanzees and gorillas. These parasites were always found by him in association with *P. vivax* and *P. malariae* forms. He concluded that anthropoid apes are as sure a source of danger to Europeans living in West Africa as are negroes.

The conclusion of Reichenow as to the identity of the parasite he found in gorillas and chimpanzees with the human parasite is interesting. The establishment of this identity would necessarily lead to important inferences. It would mean that anophelines which had fed on infected anthropoid apes could acquire salivary gland infection. Such anophelines, in parts remote from human habitation, would be capable of infecting any human being who came within their range. In this way they would constitute a permanent danger to persons employed in opening up new areas.

Reichenow's conclusion appears to us too far-reaching in view of the fact that it is based on morphological grounds only. If his conclusion is correct, it becomes difficult to understand why inoculations from human beings infected with malaria into chimpanzees should fail. The only successful inoculation of malaria from a human being into an animal is that performed by Mesnil and Roubaud (1920). These authors succeeded after several attempts in inducing a transient infection with *P. falciparum* in one of two chimpanzees. The incubation period was ten days, and the animal recovered spontaneously after another ten days. It is significant that in the two experiments using as vector *A. maculipennis* which had been infected from a case of *P. falciparum*, transmission to the chimpanzees failed entirely. This alone would suggest that *P. falciparum* is not easily transmissible to the chimpanzee, in view of the ease with which infective anophelines transmit *P. falciparum* to human beings in laboratory experiments. The failure of Mesnil and Roubaud to transmit malaria to chimpanzees by the bite of infected anophelines raises the question as to whether the *P. falciparum* forms observed by them in the chimpanzee were really due to the inoculation or were a relapse of the parasite which occurs naturally in the chimpanzee.

In order to determine whether the parasite resembling *P. falciparum* found by us in the chimpanzee was capable of infecting human beings, we performed the following experiments.

EXPERIMENTS WITH LABORATORY-BRED *A. COSTALIS*

Laboratory-bred *A. costalis* were allowed to feed on the chimpanzee on two successive nights. After a lapse of from four to fourteen days from the first feed, forty mosquitoes were dissected, and in no case was infection found either in the gut or salivary glands.

EXPERIMENTS WITH INJECTIONS OF INFECTED BLOOD

Two Europeans were given subcutaneous and intravenous injections of blood from the chimpanzee. The first subject had never had malaria, and had taken prophylactic doses of five grains of quinine bisulphate daily from January 10th to February 6th, 1922. The last dose was taken at 7 a.m. on February 6th. On February the 7th, at 5 p.m., he received subcutaneously 1 c.c. of the blood of the infected animal. An hour later slight nausea ensued, which lasted two hours. The local reaction was slight. On the 9th of February, at 10 a.m., the same subject received an injection of 0·4 c.c. of the animal's blood into his right median basilic vein. At this time the animal's infection was heavy, *i.e.*, four rings to the field (Obj. 1/12, Oc. 0, Leitz). Slight nausea followed a quarter of an hour after the injection, and lasted a few hours. The subject's blood was examined twice daily from the date of the first injection, but no parasites were found. During an observation period of twenty-eight days, no infection occurred. An interesting fact was observed, namely, that from the 12th to the 14th of February transient urticarial patches occurred, localised round the site of the first inoculation. These patches appeared and disappeared several times during the course of the day.

The second subject had previously suffered from malaria, and recently, within a year, from a *P. falciparum* infection, but had been free from relapse during the last six months. He was taking two grains of quinine bihydrochloride daily until the 7th February.

He received on February 19th, at 7 p.m., 1 c.c. of the animal's blood subcutaneously; at this time the animal's blood showed as many as nine rings to a field. No local or general reaction followed. On the 20th February, at 5 30 p.m., he received 0·2 c.c. of the animal's blood intravenously. Examination of the subject's blood before the first inoculation was negative, as were also subsequent examinations. No infection occurred during an observation period of seventeen days after the second inoculation.

The results of the above experiments lend themselves to two explanations, viz., that the parasite is *P. falciparum* which has lost its infectivity for man by passage through the chimpanzee, or that it belongs to a new species of the genus *Plasmodium*. In view of the limited number of experiments performed, we consider it premature at present to decide definitely between these two interpretations. Our experiments so far certainly do not confirm Reichenow's conclusion that chimpanzees as reservoirs of *P. falciparum* are a source of danger to Europeans in West Africa.

SUMMARY

A parasite morphologically indistinguishable from *P. falciparum* was found by us occurring naturally in a chimpanzee in Freetown, West Africa. This parasite appears to be the same as that described by Reichenow in chimpanzees and gorillas, and stated by him to be the human parasite.

Laboratory-bred *A. costalis* fed on this chimpanzee failed to become infected, but, as stated above, crescents were few and exflagellation was not observed.

We have failed to transmit the infection to two human subjects by subcutaneous and intravenous inoculation.

REFERENCES

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EXPLANATION OF PLATE IX

Forms of parasite found in the blood of the chimpanzee.

Fig. 1. *P. vivax*-like form.

Fig. 2. *P. malariae*-like form.

P. falciparum-like forms : —
    Figs. 3-10. Small rings.
    Figs. 11-14. Large rings.
    Figs. 15-17. Amoeboid forms.
    Figs. 18-20. Solid forms.
    Figs. 21-28. Crescent forms.

Pigmented leucocytes : —
    Fig. 29. Pale nucleus and finer pigment.
    Fig. 30. Dark nucleus and coarse pigment masses.

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